Spontaneous Coronary Artery Dissection of LM and LM- LAD artery in Peripartum or Postpartum Women with Acute Myocardial Infarction

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Abstract:

Spontaneous coronary artery dissection (SCAD) is a very rare condition which may result in sudden coronary occlusion, acute myocardial infarction and sudden cardiac death. It usually occurs in young women during pregnancy or postpartum period and in most cases it involves a single coronary artery. The exact etiology is unknown. The prognosis of SCAD is uncertain and optimal treatment is unknown. Early diagnosis and treatment are key for survival, and when identified early, mortality rate is reduced.

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Introduction:

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute myocardial infarction in the general population, especially in the younger patients without classic coronary artery risk factors.[1] It has been most commonly described in middle-aged otherwise healthy women in the peripartum period. Often, referred as "pregnancy associated coronary artery dissection" (PCAD). [2] Mortality rate was as high as 66%, has been reduced to 38% probably due to earlier diagnosis and intervention.[3,4]

Cases 1:

A 30 year old lady, at full term gestation complained of chest pain during the induction of labor by vaginal delivery. Induction failed and went for emergency caesarian section (C/S) under spinal anesthesia. Chest pain was overlooked by the surgeon and left untreated. Again 2 days later, she developed chest pain again while in the hospital and diagnosed as Anterior Myocardial infarction (MI) and treated conservatively. Later on, she was brought to our hospital 12 days after C/S due to ongoing chest pain. Her admission ECG showed persistent ST elevation in Anterior leads. Echocardiography revealed hypokinetic antero-septal region with LV ejection fraction (LVEF) 35%. Her coronary artery disease (CAD) risk factor was absent. She was taken to cath lab because of ongoing chest pain and revealed: Left main (LM) Stem dissection with extension beyond and distal flow compromised (Fig.1). Considering her LM-LAD dissection, patient was advised for coronary artery bypass grafting (CABG), but the patient refused. So, PCI to LM and LM to left anterior descending artery (LAD) was done. First, LM lesion was directly stented with a 3.5 x 16 mm (Promus Element) at 12-14 ATM (Fig. 2). LM-LAD lesion was then stented with a 3.0 mm x 30 mm stent (Pro-kinetic) at 10 ATM (Fig.3). Overlapping the previous stent and covering the mid LAD lesion with a 2.5 mm x 22 mm stent (Pro-Kinetic) was deployed at 12 ATM (Fig.4). LM-LAD was post dilated with a 3.5x16 mm stent balloon at 16ATM. Finally, LM ostium was post dilated at 18-20 ATM pressure. Final Cine shows TIMI III distal flow.

Case 2:

A 30 year old lady at 36wks of gestation was admitted to our hospital through cardiac OPD with the acute onset of severe chest pain. She had history of on and off chest discomfort, choking sensation around the throat for few days before

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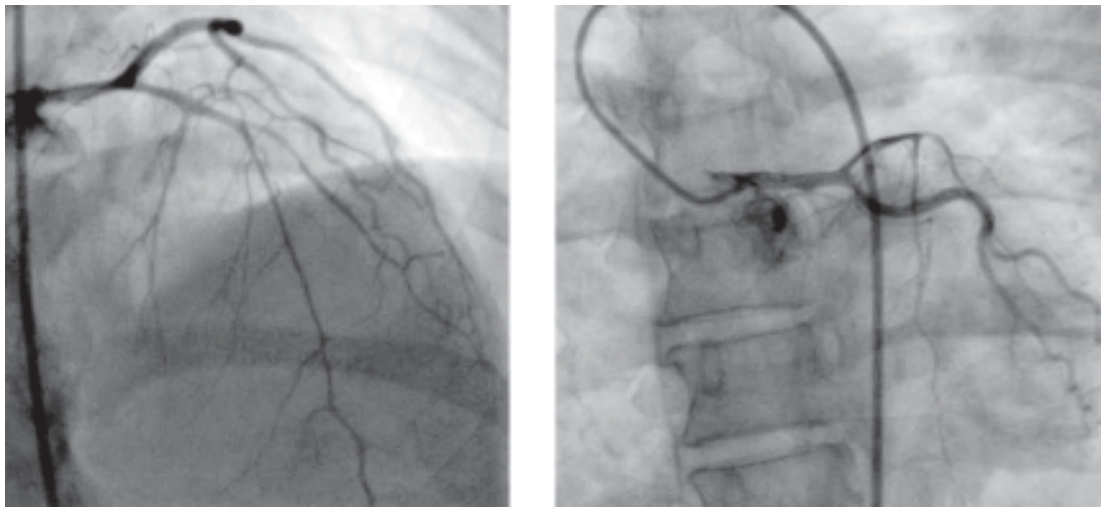

Fig.-1: *LM stem Dissection with extended beyond and distal flow compromised.*

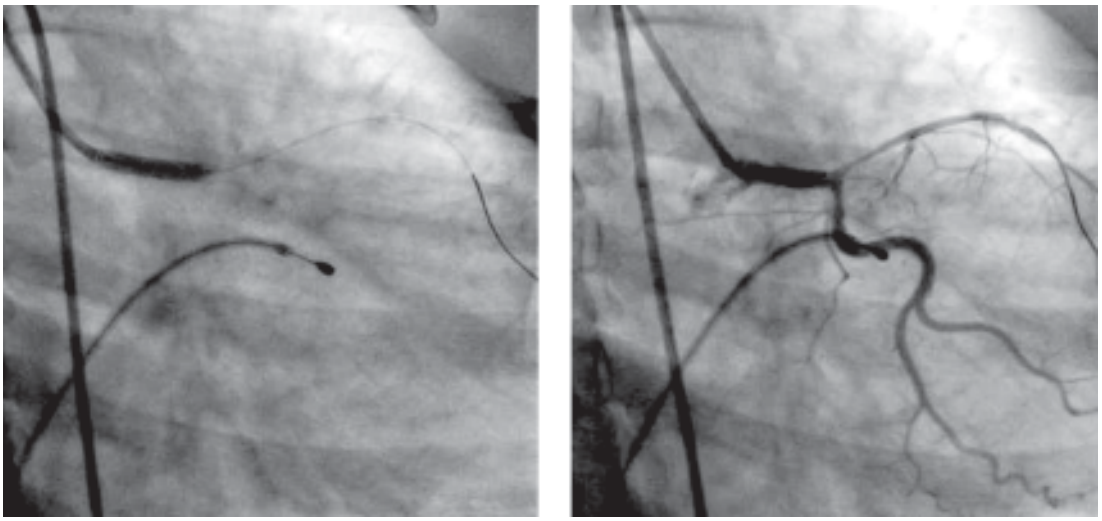

Fig.-2: *LM lesion was directly stented.*

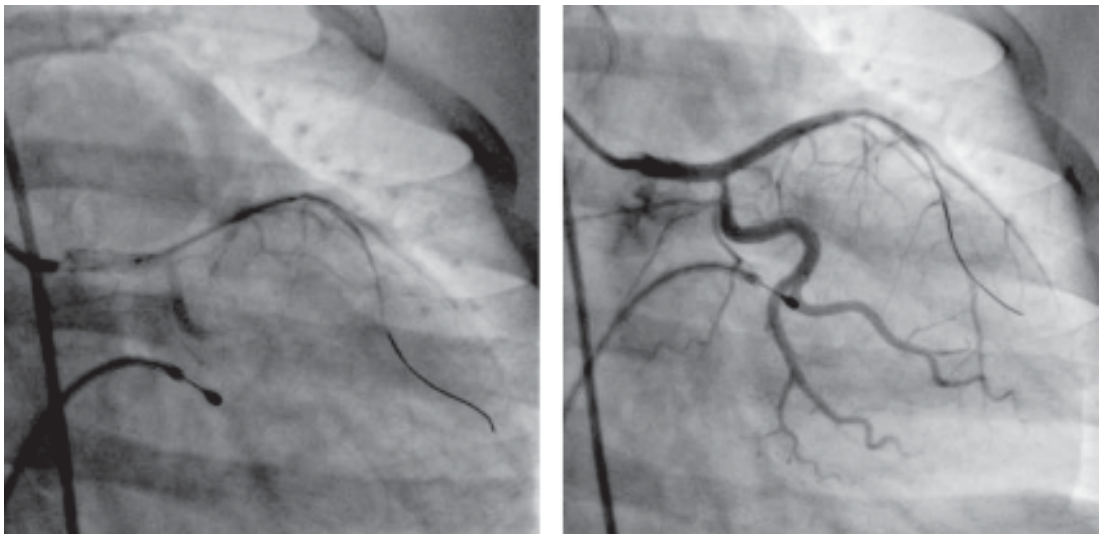

Fig.-3: *LM-LAD lesion was then stented.*

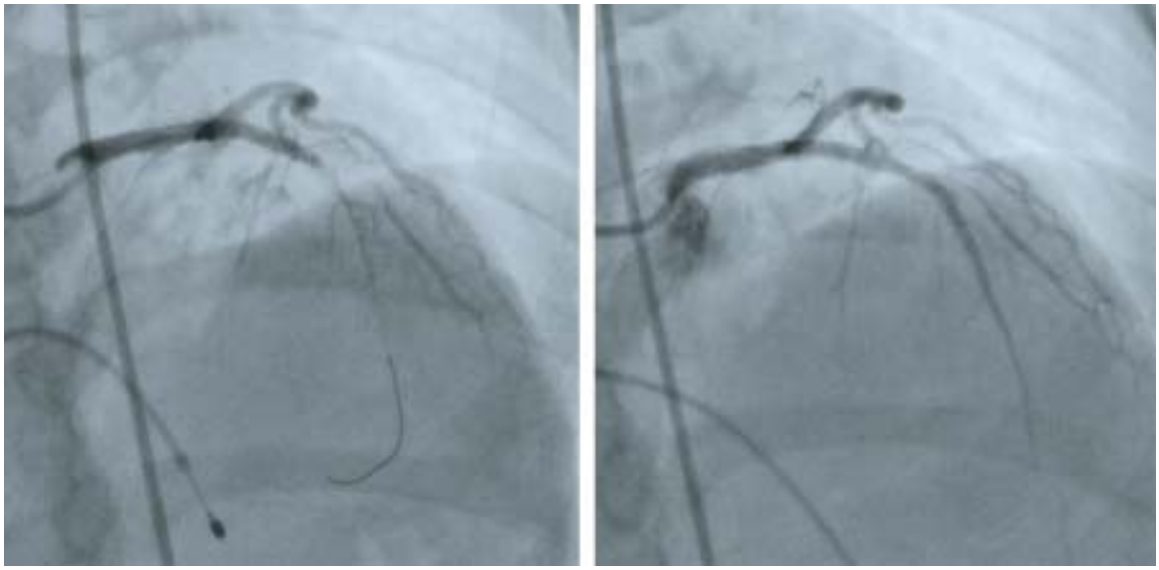

Fig.-4: *Stent deployement overlapping the previous stent and covering the mid LAD.*

referred to cardiac OPD by treating obstetrician. Her ECG showed acute ST elevation in anterior leads and shifted to coronary care unit. Echocardiography showed, hypokinetic antero-septal region with LVEF 45%.

CAD Risk factors were absent. CAG done same day with lead protection through RRA and revealed: thrombotic total occlusion of proximal LAD (Fig 5). Left circumflex (LCX) and right coronary artery (RCA) were normal. After 2 weeks, LAD lesion was stented with a 3.0 x 18 mm Resolute Integrity stent at 14-16ATM (Fig 6). Further, post dilatation was done with a 3.5 x 10 mm balloon at 14-16ATM. Final cine showed no residual plaque with TIMI – III distal run off.

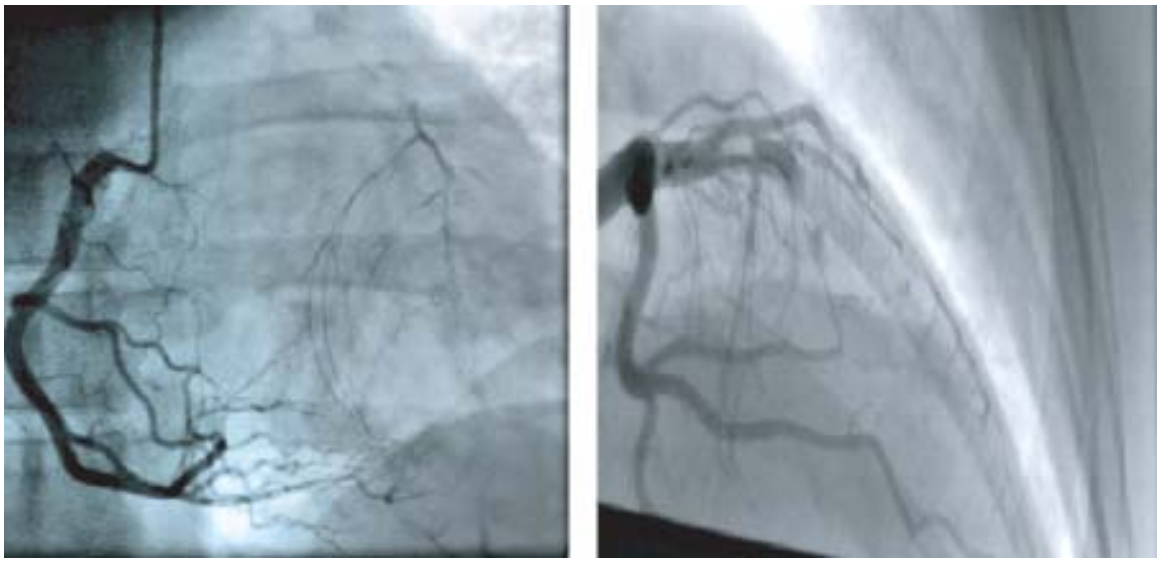

Fig.-5: *CAG revealed thrombotic total occlusion of proximal LAD. LCX and RCA were normal.*

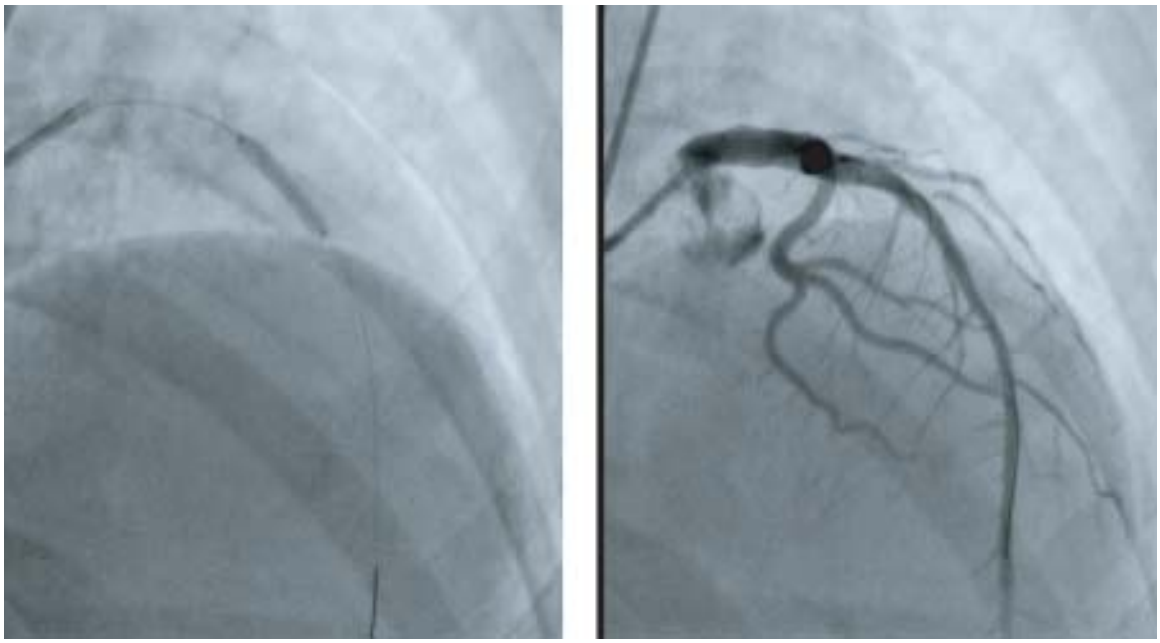

Fig-6: *Stenting of LAD after 2 weeks of conservative management.*

Discussion:

Acute myocardial infarction (AMI) is a rare and very uncommon clinical condition in women of childbearing age under the age of 40yrs.[5] This age group accounts for about 5.6% of all cases of AMI. Among them, women under 40 years account for just 0.7% of cases.[6]

However, pregnancy increases the risk of AMI 3-4 folds and occurs in about 6.2 cases per 100,000 deliveries.[5] Non-traumatic SCAD was first detected in a 42 yr old women by Pretty in 1931, in the pre-cath era by necropsy.[7] Of the cases of AMI in pregnant and postpartum women, SCAD accounted for 27% of cases compared to 1% in general population.[8] It occurs most commonly in the third trimester of pregnancy and in the early postpartum period. It involves the left main and LAD in 80% cases and the overall mortality 50% at presentation.[3] It is also not associated with the classic CAD risk factors. In women with SCAD, there is a predilection for the left coronary system (84%), in men, the right coronary artery (67%). [9] Koul et al, has reported an overall mortality of 38% mostly occurring within the first few hours from the onset of symptoms. [4]

Exact etiology remains unclear; although a significant proportion of these patients (25%) are in the puerperium. The use of oral contraceptives and the exceptional hormonal balances in the peripartum period are supposed to weaken the arterial wall and predispose it to rupture or dissection. Connective tissue diseases, cocaine abuses, systemic hypertension and intense physical exercise may be the other causes.[10] The cause of increased rates of AMI and SCAD in pregnant and post partum women probably due to hormonal and haemodynamic changes with increase cardiac output, decreased collagen production, disruption of vasa vasorum and alteration in coagulation-fibrinolysis system.[11] In about 30% SCAD, underlying cause is an atherosclerotic plaque rupture.[12] The increased density of vasa vasorum and the elevated shear stress may lead to rupture of intima and thereby creating subintimal haematoma leading to dissection.[13]

Clinical Manifestation:

The clinical manifestation of SCAD ranges from asymptomatic to acute coronary syndrome, Cardiogenic shock and sudden cardiac death.[14] The number of involving arteries, the rate of propagation of the dissection and the extent of involvement also affect the clinical manifestation.[15]

Management:

The management of the pregnant or postpartum patient depends on the haemodynamic status of the patient, viability of fetus and the extent of

myocardial damage.[16] Some of the author described that these group of patient had full recovery after conservative treatment.[17] Conservative therapy in the form of aspirin, nitrates, beta-blockers and antiplatelet has been associated with clinical and angiographic resolution. Thrombolytic therapy could lyse a compressing intramural clot, but it could also contribute to the expansion of an intramural haematoma.[9] In addition, it has been used in the past but lost favor owing to the potential risk of postpartum hemorrhage and fetal intracranial hemorrhage.[18] Percutaneous coronary interventions have also favorable outcome. Coronary stenting of SCAD was first reported in 1996 and become the treatment of choice for preventing further propagation of dissection. [19] Porto et al reported the first case of drug eluting stent (DES) implantation in a patient with SCAD.[20] At 2 years follow-up the patient was asymptomatic with patient stent and no evidence of neointimal hyperplasia at angiography and IVUS studies.[21] PCI should be first therapeutic choice in single vessel dissection with persistent impairment of blood flow and ongoing symptoms of ischemia. Simple balloon dilatation should be avoided, whereas coronary stenting has been successfully selected cases where the true and false lumen were clearly distinguishable and the dissection not involve a long segment [22] CABG should be the choice of treatment in cases with LM stem involvement, multivessel disease and ongoing ischemia with refractory to medical or interventional therapy.[23]

Conclusion:

SCAD is a rare cause of acute coronary syndromes. It should always be suspected as a cause of acute coronary syndromes in young female patient especially during pregnancy. PCI in single or multivessel dissection with or without LM stem involvement could be an effective alternative to CABG in patient with SCAD in the peripartum or postpartum period in our population.

Conflict of Interest - None.

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